Atrioventricular Junctional Premature and Escape Beats with Altered QRS and Fusion

By Albert D. Kistin, M.D.

THIS STUDY presents electrocardiographic data from six patients which indicate that (1) atrioventricular (A-V) junctional premature and escape beats may give rise to QRS complexes which differ in configuration from the QRS of sinus origin and which cannot be explained by intraventricular block or aberrant conduction during incomplete recovery, (2) QRS complexes occur which seem to be produced by fusion of ventricular activation which originates partly from the A-V junctional impulse and partly from the sinus impulse, and (3) these two facts may be explained by conduction of the A-V impulse into the ventricles by some pathway other than the usual A-V pathway. The first and third points were first suggested by Kaufmann and Rothberger,1 and in a footnote to one of their tables they described QRS complexes which were intermediate in form between the QRS complexes of sinus origin and those of ectopic origin, although they did not illustrate or discuss these.

Kaufmann and Rothberger1 identified premature A-V junctional beats in venous pulse tracings by c-to-a intervals which they considered too brief to be explained by retrograde conduction from a ventricular focus. They found that these beats had a consistent abnormal QRS configuration in electrocardiograms which in their opinion could not be explained by ordinary aberrant conduction during incomplete recovery, because early postextrasystolic QRS complexes had normal configuration. Their hypothesis was that the A-V junctional impulse might be conducted to the ventricles by selected fibers of the bundle of His.

Others2–7 have made the interpretation that escape beats of A-V junctional origin may produce a QRS which differs in configuration from the QRS of sinus origin and have expressed the opinion that this occurs frequently.3,7 In these cases, however, the possibility cannot be excluded that the illustrated ectopic beats are ventricular rather than A-V junctional in origin. Pick8 suggested that the A-V impulse may be conducted by way of the fibers first described by Mahaim8 and later by others,9–11 which go directly from the A-V node, the A-V bundle, or the left bundle branch to septal myocardium. Massie and Walsh,5 Goodman and Pick,6 and Walsh7 described what they believed to be fusion QRS complexes which originated partly from an A-V junctional escape impulse and partly from the sinus impulse.

Methods and Cases Studied

The A-V junctional beats (figs. 1 to 8) in this study were identified in simultaneous vector12,13 and esophageal leads14 by R-to-P intervals which were too brief to be explained by conduction from peripheral ventricular foci to the atria, and in one case by brief P-to-R intervals, too brief to be explained by atrioventricular conduction. In four cases the A-V junctional beats were premature (figs. 1, 2, 5, and 6), in one case both premature and escape beats (figs. 3 and 7), and in one case only escape beats (figs. 4 and 8). The beats labeled J were recorded in all cases in repeated tracings of simultaneous vector leads over periods of months to more than a year and J maintained about the same configuration. The simultaneous vector leads were used to reduce the possibility that a false brief R-to-P interval might sometimes be produced because the initial part of ventricular activity might not be recorded in a given lead.15,16 The illustrations prove the indispensability of the esophageal leads; early retrograde P waves which

From the Cardiopulmonary Laboratory, Beckley Appalachian Regional Hospital, Beckley, West Virginia, and the Department of Medicine, George Washington University School of Medicine, Washington, D.C.

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Case 1. Four parts of a tracing consisting of simultaneous X, Y, and Z leads (Schmitt and Simonson12) and, in the upper groups, a bipolar esophageal lead 41 cm from the nares (BE 41). (Upper left) J occurs immediately after P and is not followed by P'. (Upper right) J is followed by P' which is interpreted as produced by retrograde activation of the atria. By comparison of the upper left and the upper right tracings, it is possible to measure the interval from the onset of the QRS of J to the onset of P'. Because of the brief interval, too brief to be explained by retrograde conduction from a ventricular focus, J is interpreted as originating in the A-V junction. (Lower left) Three atrial premature systoles produce QRS complexes like those of sinus origin. It is unlikely that the QRS configuration of J may be explained by aberrant conduction during incomplete recovery because (1) the QRS produced by the first atrial premature systole occurs earlier in the cycle than J, and the preceding cycle, R-R, is about the same as the cycle which precedes J (upper left) and (2) the QRS of J maintains the same configuration late in the cycle (upper left) and also earlier (upper right). (Lower right) The configuration of QRS of F is intermediate between that of the QRS of sinus origin and that of J, and it occurs at an interval after sinus P which is consistent with the interpretation that it is produced by ventricular activation partly from an impulse of sinus origin and partly from an impulse of A-V junctional origin. (Reproduced by permission of Grune & Stratton, Inc.19)
were played back at 100 mm per second for more accurate measurements of intervals. The earliest discernible onsets of QRS and P in any lead were used for the measurements of intervals, and the earliest onset and latest termination in any lead were used for the measurement of duration of QRS. In the illustrations some of the rapid deflections are retouched. Figures 3 to 5 and 7 present tracings recorded at a paper speed of 25 mm per second. Figures 1, 2, 6, and 8 with time indices present tracings recorded at a paper speed of 50 mm per second.

Tracings from three of the patients (cases 1, 3, and 4) were previously used in papers on esophageal leads and the differential diagnosis of cardiac arrhythmias.18, 19

Case 1

The patient was a 59-year-old coal miner with bronchial asthma and pulmonary emphysema. The electrocardiograms sometimes showed a P-pulmonary pattern. He did not receive digitalis. He died of metastatic carcinoma 3 months after the date of the tracings of figure 1; the primary site was not found.

Case 2

A 53-year-old former coal miner had complicated coal workers’ pneumoconiosis and pulmonary emphysema. The electrocardiogram was consistent with right ventricular hypertrophy. He did not receive digitalis.

Case 3

The patient, a 62-year-old former coal miner, had hypertensive heart disease, episodes of congestive heart failure, and radiological evidence of cardiac enlargement. The electrocardiogram was consistent with left ventricular hypertrophy. The blood serological tests were positive for syphilis. He had not had digitalis for a month or more when the tracings of figure 3 were recorded. He was taking digitalis when the tracing of figure 7 was recorded.

Case 4

A 39-year-old coal miner with scoliosis had no clinical evidence of heart disease, and his electrocardiogram was considered normal. He did not receive digitalis.

Case 5

A 50-year-old rigger with eczema of the hands and feet presented no evidence of heart disease, and he did not receive digitalis.

Case 6

The patient was a 68-year-old former coal miner with coronary arteriosclerotic and hyper-

Figure 2

Case 2. Three parts of a tracing consisting of simultaneous X, Y, and Z leads (Schmitt and Simonson18) and in the upper group, a bipolar esophageal lead 39 cm from the nares (BE 39). Interpretation of P', F, and the maintenance of the QRS form of J in different parts of the cycle as in figure 1. In the middle tracings J occurs a little earlier than in the lower tracings and there is no fusion; such data permit an estimate of the time it takes the ectopic impulse to reach and activate the first part of myocardium usually activated by the sinus impulse (discussed in text).
Case 3. Four parts of a tracing consisting of simultaneous X, Y, and Z leads (Frank\textsuperscript{13}) and in the upper group a bipolar esophageal lead 36 cm from the nares (BE 36). J occurs both as premature beats in the upper and lower right groups and as escape beats in the lower left group. Interpretation of J, P', and F as in figure 1. The QRS form of J is about the same in different parts of the cycle (upper left and lower) but is altered somewhat, especially in the X lead, when it occurs earlier in the cycle (upper right group), possibly because of occurrence during incomplete recovery.

tensive heart disease, cardiac enlargement, and pulmonary emphysema. The electrocardiograms were interpreted as showing an old diaphragmatic and lateral myocardial infarct. He did not receive digitalis.

**Observations**

In each case, tracings were obtained that show in the same strip atrial premature systoles as well as the beats labeled J (figs. 1 and 6). The QRS produced by the atrial premature systoles has a configuration like that of the QRS of sinus origin, and it occurs at an interval from the preceding QRS as brief or briefer than the interval between J and the preceding QRS. The duration of the cycle which precedes the QRS of the
Figure 4

Case 4. Simultaneous leads I, II, V_1, and V_5, and in the upper group a bipolar esophageal lead 34 cm from the nares (BE 34). Interpretation of J, P', and F as in figure 1. P' sometimes immediately precedes and sometimes immediately follows the QRS of J, evidence that J originates in the A-V junction. (Reproduced by permission of Grune & Stratton, Inc.)

atrial premature systole—preceding R to R—is as long or longer than the cycle which precedes J.

QRS-to-P' and P'-to-QRS Intervals

In each case the onset of the QRS of J is followed often, and in one case always,
after a brief interval by a large deflection in the esophageal leads (P' in the illustrations) which differs in configuration from the sinus P wave, and in some esophageal leads is more or less opposite in direction (figs. 1 to 7). When J is premature, P' occurs earlier than the next expected sinus P wave (fig. 2) or about the time that the sinus P wave is expected and prevents the occurrence of this sinus P wave (figs. 1 to 3 and 5 to 7).

Although P' often occurs so early as to be superimposed on the QRS of J, the delineation of the configuration of P' is possible, and the measurement of QRS to P' intervals is possible for several reasons. In five of the cases, J is sometimes not followed by P' so that the configuration of P' may be determined when it is superimposed on QRS (figs. 1 and 3 to 5). In one case in which P' is always superimposed on the QRS of J, P' occurs also after probable ventricular premature systoles and is well separated from them so that the configuration of P' can be determined (fig. 6). In the one case in which J occurs only as escape beats, P' sometimes precedes the QRS of J and sometimes follows it (fig. 4).

The briefest QRS to P' intervals of J in cases 1, 2, 3, 5, and 6 are 0.04, 0.05, 0.04, 0.05, and 0.06 sec. In case 4 there are P'-to-QRS intervals as brief as 0.05 sec as well as brief QRS-to-P' intervals (fig. 4).
Case 6. (Upper) Simultaneous X, Y, and Z leads (Frank*3) and a bipolar esophageal lead 39 cm from the nares (BE 39). (Lower) Simultaneous X lead and bipolar esophageal leads, 35, 37, and 39 cm from the nares (BE 35, 37, and 39). Interpretation of J and P' as in figure 1. V is probably a ventricular ectopic beat with a QRS-to-P' interval consistent with that interpretation; it occurs later in the cycle than J and after a briefer preceding cycle, so that it is unlikely that its longer QRS-P' interval compared with J may be explained by conduction during incomplete recovery. The significance of the timing of the QRS of the atrial premature systole at the end of the upper tracings, and the configuration of P' after J and V are discussed in the text.
Case 3. Simultaneous X, Y, and Z (Frank) and bipolar esophageal leads 32, 34, and 36 cm from the nares (BE 32, 34, and 36). Interpretation of J and P' as in figure 1. V is probably a ventricular ectopic beat with a QRS-to-P' interval consistent with that interpretation; it occurs later in the cycle than the last J of the tracing and after a brief preceding cycle, so that it is unlikely that its longer QRS-P' interval compared with J may be explained by conduction during incomplete recovery. J occurs both as premature and escape beats. The QRS of the last J is altered, especially in the Z lead, probably because it occurs earlier in the cycle and because of aberrant conduction during incomplete recovery, even though the basic configuration of J cannot be explained by such aberrant conduction (discussed in text).

Intermediate QRS Complexes

QRS complexes whose configuration is intermediate between that of the QRS of sinus origin and that of J are illustrated in figures 1 to 5 and 8. The interval from the preceding sinus P wave to the intermediate QRS is always slightly less than or about equal to the interval from a sinus P wave to a QRS which is entirely of sinus origin. The difference between the intervals sinus P-to-usual QRS and sinus P-to-intermediate QRS is in no case more than 0.02 to 0.03 sec. In one case the onset of the intermediate QRS sometimes resembles the onset of the QRS of sinus origin and at other times resembles the onset of the QRS of J (fig. 8).
Figure 8

Case 4. Simultaneous X, Y, and Z leads (Frank12). Other simultaneous tracings showed that J was the same as that shown in other leads in figure 4. Interpretation of J and F as in figure 1. The QRS of sinus origin has a small Q in the X lead but not in the Y lead. The QRS of J has a small Q in the Y lead but not in the X lead. F in the upper tracings has a small Q in the X lead but not in the Y lead; here the impulse of sinus origin activated the ventricles first. F in the lower tracings has a small Q in the Y lead but not in the X lead; here the ectopic impulse of A-V junctional origin activated the ventricles first. Such variation fits the concept of fusion.

Discussion

Evidence for an A-V Junctional Focus

P' is interpreted as being produced by retrograde conduction to the atria from an A-V junctional focus and in cases 3 and 6 also from a ventricular focus, because of its association with these ectopic beats, its configuration in esophageal leads, and because it sometimes precedes and always prevents the occurrence of the next expected sinus P wave. The brief QRS-to-P' intervals associated with J suggest that J is probably derived from a focus in the A-V junction; the impulse is conducted at about the same time to both the atria and the ventricles. When P' sometimes precedes and sometimes follows QRS as in figure 4, the evidence for an A-V junctional origin of J seems convincing. The conduction time from a peripheral ventricular focus to the atria in man may be taken roughly as at least 0.10 sec on the basis of estimates that have been made of the conduction time from a ventricular focus20, 21 the estimates of the time it takes an impulse to traverse the A-V node,22 and the experimental evidence that the velocity of conduction through the A-V junction is about the same in the forward and retrograde directions.23

In a limited number of observations the QRS-to-retrograde P interval of ventricular premature systoles produced by a catheter in the right ventricle was at least 0.11 sec.19

A brief QRS-to-retrograde P interval might conceivably be produced by rapid retrograde conduction from a ventricular focus by way of something like a Wolff-Parkinson-White pathway, but in repeated tracings obtained over periods of months to years, there was never evidence of the Wolff-Parkinson-White syndrome in the patients studied. In two of the cases (figs. 6 and 7) the configurations of retrograde P waves in three simultaneous esophageal leads could be compared after J and after a probable ventricular premature beat. They were the same, indicating that the pathway of retrograde conduction was the same. The interval between the QRS of probable ectopic ventricular origin and P' is what would be expected with conduction from

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a ventricular focus through the A-V node. This suggests that the retrograde conduction from J also occurred by way of the A-V node, and that the focus for J was located somewhere in this region. Possibly additional evidence that in some of the cases, J is not too far removed from the usual A-V pathway is the fact that QRS in some leads differs only slightly from the QRS of sinus origin (figs. 3 and 5).

**Evidence for an Unusual A-V Pathway**

The abnormal configuration of the QRS of J cannot be explained by intraventricular block or aberrant conduction during incomplete recovery. Aberrant conduction during incomplete recovery is more likely when an impulse of supraventricular origin occurs early in the cycle or after a long preceding cycle,\(^\text{24, 25}\) or both. If in a given tracing under apparently similar conditions normal QRS complexes occur equally early in the cycle or earlier and after equally long or longer preceding cycles, then it is unlikely that the abnormal QRS may be explained by aberrant conduction during incomplete recovery. Such evidence is present in all six cases and is illustrated in figures 1 and 6.

Sometimes J maintains about the same configuration in widely different parts of the cycle, even quite late in the cycle (figs. 1 to 3, and 5), and in these cases this is additional evidence that the QRS configuration of J is probably not the result of aberrant conduction during incomplete recovery. When the normal QRS produced by the atrial premature systole occurs earlier in the cycle than J as in figure 1, the hypothetical possibility of a supernormal phase might be considered as the explanation for the normal QRS. In cases 3 and 6 (fig. 6), however, the normal QRS of atrial premature systoles occurred at the same time as J and after cycles of the same duration, and in these cases the evidence seems conclusive that J cannot be the result of aberrant conduction during incomplete recovery. Even though the basic configuration of the QRS of J cannot be explained by aberrant conduction during incomplete recovery, J may, of course, occur during incomplete recovery, and its QRS configuration may be altered further for that reason (fig. 7).

The intermediate QRS complexes (figs. 1 to 5, and 8) have the characteristics of fusion or combination complexes,\(^\text{26-28}\) and their timing with respect to the preceding sinus P wave satisfies the conditions for fusion, in this instance fusion of activation of the ventricles partly by the impulse of sinus origin and partly by the impulse of A-V junctional origin. The simplest explanation is that the sinus impulse and the A-V junctional impulse traverse different paths. Either the pathway of selected fibers within the bundle of His suggested by Kaufmann and Rothberger\(^\text{1}\) or the pathway of paraspecific fibers suggested by Pick\(^\text{3}\) would fit the observations.

Since the quantity, the usual PR interval minus the interval from sinus P-to-intermediate QRS, ranges from zero to not more than about 0.03 sec, this suggests that the conduction time from the A-V focus by way of the unusual pathway to the first part of myocardium ordinarily activated by the usual A-V pathway is not more than about 0.03 sec. This interval is briefer than that observed with epicardial ventricular foci\(^\text{21}\) or in the Wolff-Parkinson-White syndrome\(^\text{29}\) and seems consistent with the concepts of either Kaufmann and Rothberger\(^\text{1}\) or Pick.\(^\text{3}\) In case 4 the fusion concept is confirmed by the fact that sometimes the fusion seems to start with ventricular activation by way of the sinus impulse and sometimes with activation by way of the A-V junctional impulse (fig. 8).

Explanations involving selective activation of some of the fibers of the A-V bundle in one manner or another were used by Fenichel,\(^\text{30}\) Fulchiero,\(^\text{31}\) Geraudel,\(^\text{32}\) and Prinzmetal and his associates\(^\text{33}\) for the Wolff-Parkinson-White syndrome. Rakita and his associates\(^\text{34}\) used a similar explanation for the production of bizarre QRS complexes on stimulation of the A-V junction. Singer and his associates\(^\text{35}\) suggested that low membrane potential due to slow diastolic depolarization rather than different pathways may explain the altered configuration of escape beats in experimental studies. Such a mechanism does
not seem to explain the fusion beats in the clinical tracings nor the observation in two cases that normal QRS complexes which result from atrial premature systoles may occur in the same part of the cycle as J, and after preceding cycles which are the same (fig. 6).

**Implications for Differential Diagnosis of Cardiac Arrhythmias**

Fusion complexes heretofore have been considered among the best evidence for the ventricular origin of ectopic beats. If beats of A-V junctional origin may be conducted by way of an unusual pathway and therefore may be associated with fusion complexes, and if A-V junctional beats may be associated with abnormal QRS complexes with or without intraventricular block and aberrant conduction during incomplete recovery, then the differential diagnosis between supraventricular and ventricular arrhythmias becomes even more difficult than has been supposed. An important consideration will be the frequency with which such beats occur, and this requires further study.

Goodman and Pick suggested that fusion may still be a good criterion for a ventricular focus if the QRS duration of the ectopic beat exceeds 0.12 sec, but the QRS duration does not seem to be completely reliable. Ventricular ectopic impulses which originate in the interventricular septum may give rise to a QRS of normal duration. In the tracing from which figure 3 was made, the average duration of the QRS of sinus origin is 0.12 sec, and the average duration of the QRS of a number of premature J beats which are not followed by retrograde P waves is 0.14 sec. This study shows that the beats under discussion occur as premature beats as well as escape beats, and this introduces another complication regarding QRS duration; the ectopic beats may occur during incomplete recovery, their QRS duration may be prolonged for this reason (fig. 7), and it is to be expected that QRS duration of more than 0.12 sec may occur.

**Summary**

The interval between an ectopic QRS and a retrograde P wave measured in simultaneous esophageal and other leads may help to identify beats which originate in the atrioventricular (A-V) junction. In six cases beats which seem to originate in the A-V junction are associated with a QRS which differs in configuration from the QRS of sinus origin, and in each case atrial premature systoles which give rise to normal QRS complexes occur in tracings which contain the A-V junctional beats also. Comparison of the QRS of the atrial premature systoles with the QRS of the A-V junctional beats with respect to the time of occurrence in the cardiac cycle and the duration of the preceding cycle makes it seem unlikely that the altered QRS of the A-V junctional beats can be explained by aberrant conduction during incomplete recovery. In five of the cases, fusion QRS complexes occur, suggesting activation of the ventricles partly by an impulse of sinus origin and partly by the impulse of A-V junctional origin. These observations support the interpretation previously made by others that impulses of A-V junctional origin may reach the ventricles by pathways other than the usual A-V pathway. If such beats as are described here occur at all frequently, then the diagnostic value of fusion, heretofore considered an almost conclusive criterion of a ventricular focus, may be impaired.

**References**


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ALBERT D. KISTIN

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